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ORIGINAL RESEARCH

A Prospective Study of Spectrum of Depressed Fractures of Skull and its Surgical Outcome

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ABSTRACT

Background: Depressed skull fracture is a very serious type of trauma occurring in 11% of severe head injuries, and there is a consistent association between the presence of cranial fracture and higher incidence of intracranial lesions, neurological deficit, and poor outcome. Depressed cranial fractures have to be treated aggressively because of their association with infection and late epilepsy.

Objectives: To study the clinical profile and surgical outcome of patients with depressed cranial fractures.

Materials and methods: A case series study of 44 patients with depressed fracture was conducted in a tertiary care hospital setting at the Department of Neurosurgery, Vijayanagara Institute of Medical Sciences, Bellary, Karnataka, during the period from June 2013 to January 2015. Among the selected patients, the clinical profile, radiological profile, and surgical interventions were undertaken and the outcomes were noted. Appropriate descriptive statistics were used to analyze the findings and to draw inferences.

Results: There were 30 males and 14 females. The mean age of the patients was 26.95 ± 14.87 years (6–65 years). The common cause of depressed fracture was road traffic accident (45%) and assault (40.9%); 63% of them had compound type and half of the fractures were located in the frontal region. Common associated injuries were extradural hematoma (50%) followed by dural tear (27.3%). Common complications were wound infection (9.1%) and cerebrospinal fluid leak (9.1%).

Conclusion: The management of depressed fractures should be individualized depending on factors like the degree of depression, communication with the exterior, neurological deficit and presence of associated injuries.

Keywords: Cerebrospinal fluid leak, Depressed cranial fractures, Surgical outcome, Wound infection.

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INTRODUCTION

Depressed skull fracture is a very serious type of trauma occurring in 11% of severe head injuries,[1] and there is a consistent association between the presence of cranial fracture and higher incidence of intracranial lesions, neurological deficit, and poor outcome.[2] Skull fractures are classified in three ways – by pattern (linear, comminuted, depressed), by anatomic location (vault convexity, base), and by skin integrity (open, closed). In a depressed skull fracture, the greatest depression can occur at the interface of the fracture and the intact skull or near the center of the fracture if several fragments are displaced inward.[3] Complex depressed fractures are those in which the duramater is torn. Depressed skull fractures may require surgery to lift the bones off the brain if they are causing pressure on it. The treatment of depressed skull fracture depends on the degree of depression, communication with the exterior, and neurological deficit. Indications of surgery in depressed skull fracture are compound depressed fracture, focal neurological sign, cerebrospinal fluid (CSF) leak, depression more than the inner table of nondepressed bone, other associated lesions like extradural hematoma EDH, and cosmetic purpose, e.g., over the forehead.[4]

MATERIALS AND METHODS

A case series study of 44 patients with depressed fracture was studied in a tertiary care hospital setting at the Department of Neurosurgery, Vijayanagara Institute of Medical Sciences, Bellary, Karnataka, during the period from June 2013 to January 2015. All patients admitted with depressed skull fracture and who underwent operative treatment were included in the study. Among the selected patients, the clinical profile, radiological profile, and surgical interventions were undertaken and the outcomes were noted. After initial clinical assessment, computed tomography (CT) scan was done in all the patients to assess the position, extent, and number of fractures as well as the presence and depth of depression. The scan

also helped in the assessment of the underlying brain for contusion or hematoma, small bone fragments, or foreign bodies, as well as other traumatic intracranial pathology.

Surgical interventions comprised of elevation of depressed bone fragment, removal of in-driven bone fragment, repair of dural tear, evacuation of hematoma, hemostasis, debridement of wound margin, and primary repair. In cases of in-driven bone fragment, removal of bone fragment was attempted by burr hole craniotomy. All the patients were given prophylactic antibiotics and anticonvulsants. All the patients were followed up at least up to 3 months postoperatively for the presence of complications and neurological deficits. Appropriate descriptive statistics were used to analyze the findings and to draw the inferences.

The study was given ethical approval by the Ethical Review Committee of Vijayanagara Institute of Medical Sciences. All ethical requirements including confidentiality of identity, responses, and informed consent were stringently ensured throughout the project.

RESULTS

A total of 44 patients who were admitted with depressed skull fracture and who underwent operative treatment were included in the study, wherein there were 30 males and 14 females. The mean age of patients was 26.95 ± 14.87 years (6–65 years). More than half of the patients were in the age group of 15 to 35 years (Table 1).

Table 1: Age- and sex-wise distribution of the patients

Variable		*No. of patients*	*Percentage*
Age group			
	< 15 years	10	22.7
	15–35 years	24	54.5
	36–65 years	10	22.7
	Total	44	100
Mean ± SD	26.95 ± 14.87		
Sex			
	Male	30	68.2
	Female	14	31.8
	Total	44	100

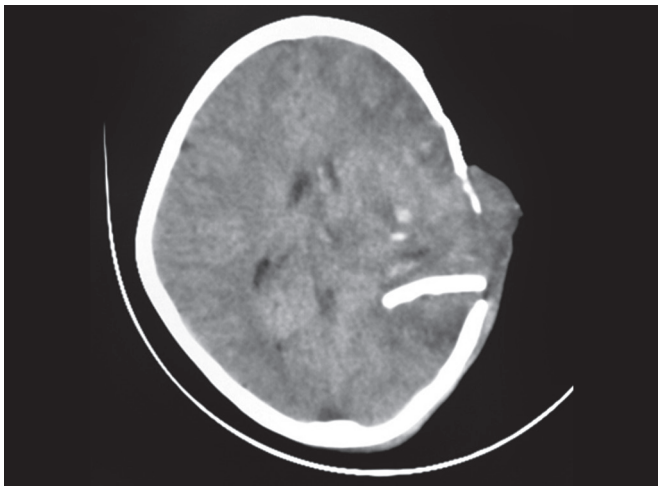

Fig. 1: Left parietal depressed fracture with in-driven bone fragment with contusion

In the present series, the common causes for depressed skull fractures were road traffic accidents (45.5%) and assault (40.9%); other causes were fall from the same level, alcohol induced, and fall of a heavy object. Nearly two-third of the cases were having a compound (open) type (63.6%) (Fig. 1) of depressed fracture, and the remaining had simple (close) type of depressed fracture (Table 2).

At the time of admission, 12 (27.3%) patients had Glasgow coma scale (GCS) of 3 to 8, 28 (63.6%) had GCS of 9 to 12, and the remainder of the patients had normal level of consciousness. The common location of depressed skull fracture was the frontal region of the skull (63.6%) followed by parietal region (27.3%); other less-common regions were temporal (4.5%) and occipital (4.5%) regions of the skull. Depressed skull fractures are associated with intracranial injuries; in the present series, half of the patients had associated extradural hematoma followed by dural tear (27.3%) (Fig. 2), in-driven bone fragment (18.2%) (Fig. 3), brain contusion (13.6%) (Fig. 4), and subdural hematoma (Fig. 5), and pneumocephalus was noted in two cases (Fig. 6, Table 2).

Surgical management of the depressed fractures was done under the cover of prophylactic antibiotics and

Table 2: Clinical profile of the patients with depressed fractures (n = 22)

Variable		*No. of patients*	*Percentage*
Etiology			
	Road traffic accident	20	45.5
	Assault	18	40.9
	Others	6	13.6
Type of fracture			
	Compound	28	63.6
	Simple	16	36.4
Level of consciousness			
	GCS: 3–8	12	27.3
	GCS: 9–12	28	63.6
	GCS: 13–15	4	9.1
Location			
	Frontal	22	50.0
	Frontoparietal	6	13.6
	Parietal	12	27.3
	Temporal	2	4.5
	Occipital	2	4.5
Associated injuries			
	Dural tear	12	27.3
	EDH	22	50.0
	SDH	2	4.5
	Brain contusion	6	13.6
	In-driven bone fragment	8	18.2
	Pneumocephalus	2	4.5

GCS: Glasgow Coma Scale; EDH: Extradural hematoma; SDH: Subdural hematoma

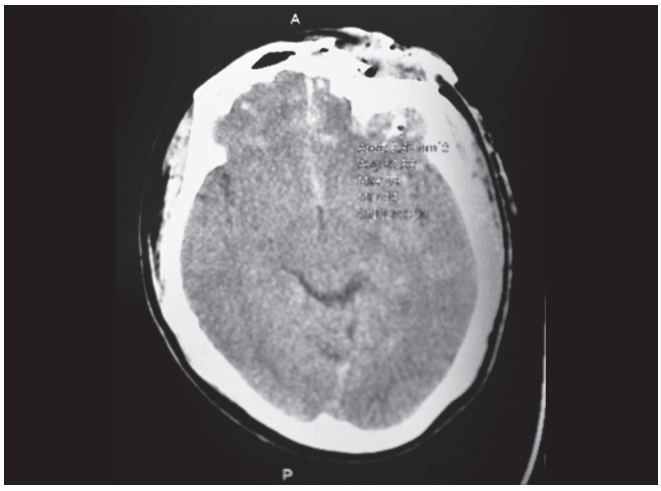

Fig. 2: Left frontal depressed fracture with multiple contusions

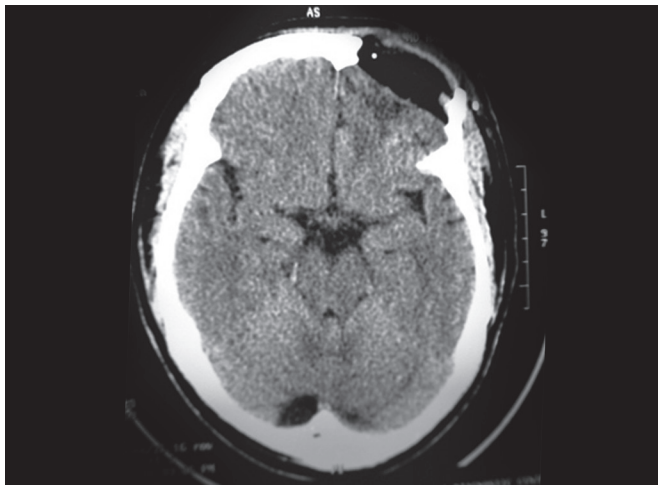

Fig. 3: Left frontal depressed fracture with pneumocephalus

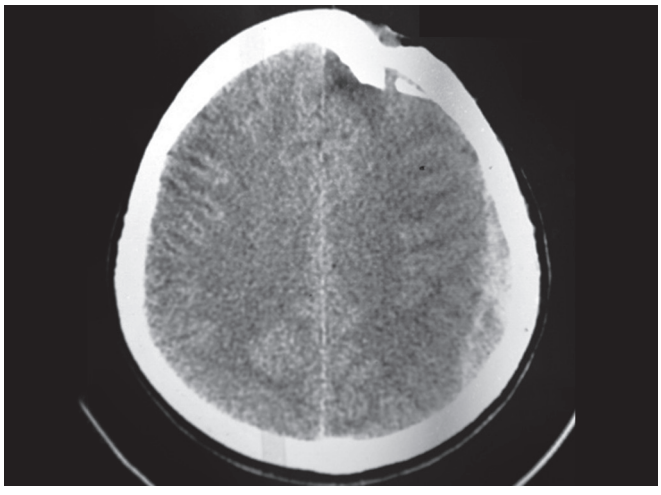

Fig. 4: Left frontal depressed fracture with subdural hematoma

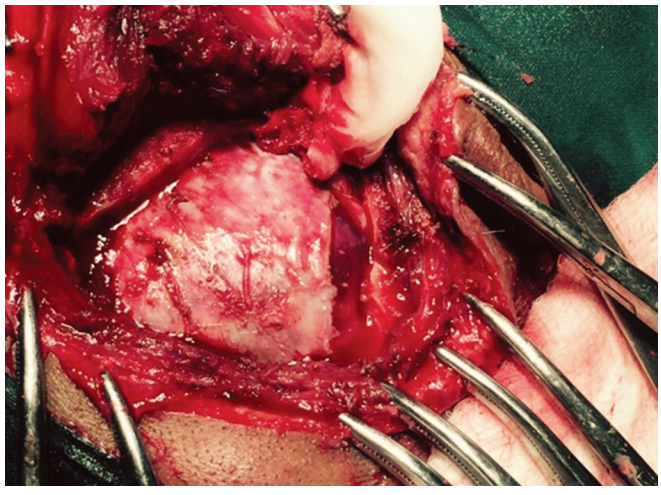

Fig. 5: Right frontal depressed fracture with associated dural tear

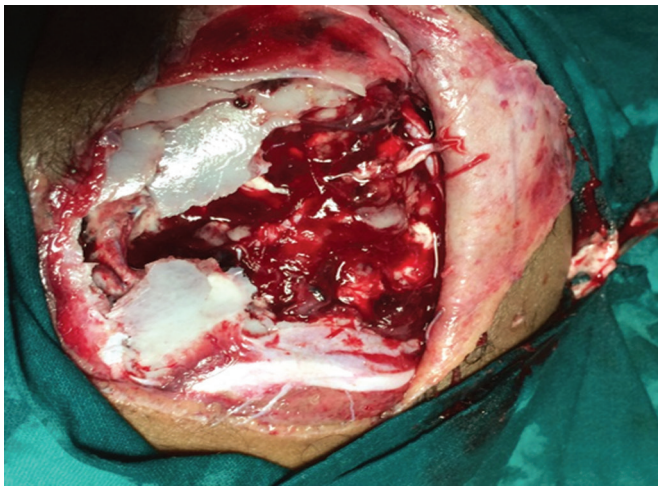

Fig. 6: Right comminuted depressed fracture in a 3-year-old child with underlying contusion and brain laceration

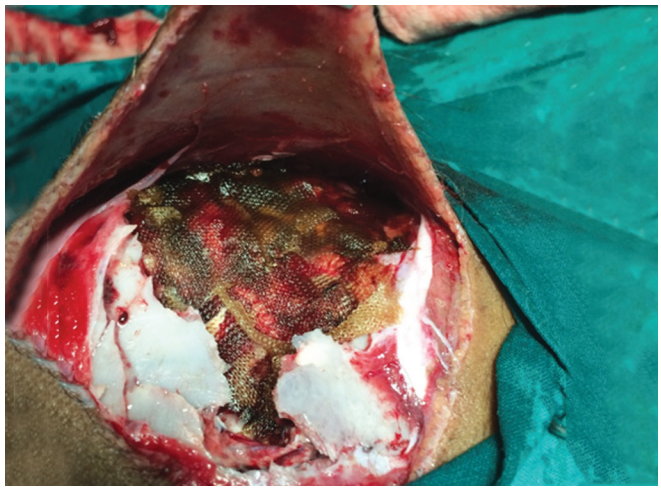

Fig. 7: The same child (Fig. 6) after achieving hemostasis and repair

anticonvulsants by elevation of depressed bone fragment, removal of in-driven bone fragment, repair of dural tear, evacuation of hematoma, hemostasis, debridement of wound margin, and primary repair (Fig. 7).

All the patients were followed up postoperatively for complications; the common complications were wound infection (11.3%) and CSF leak (9.1%), which were managed conservatively. Other complications included two cases of meningitis and one case of pseudomeningocele (Table 3). After follow-up of 3 months, three cases were having focal neurological deficits.

Table 3: Postoperative complications

Complications	*No. of patients*	*Percentage*
Wound infection	5	11.3
Meningitis	2	4.5
CSF leak	4	9.1
Focal neurological deficits	3	6.8
Pseudomeningocele	1	2.3
No complications	32	72.7
Total	44	100

DISCUSSION

A skull fracture is a break in one or more of the bones in the skull caused by head injury. These skull fractures are serious only when they affect the brain directly or indirectly. However, in the minds of the public, fractures of the skull carry an impression of severe injury with grave prognosis.[4] A force severe enough to fracture the skull my expend itself in the skull or may spread to produce brain damage as well. Broken fragments of skull can lacerate or bruise the brain or damage the blood vessels.[5]

Fractures of the skull can be comminuted, depressed, linear, or diastatic.[4] Comminuted skull fractures are those in which a bone is shattered into many pieces; it can result in bits of bone being driven into the brain, lacerating it.[6] Depressed skull fractures result from a high-energy direct blow to a small surface area of the skull with a blunt object. Comminution of fragments starts from the point of maximum impact and spreads centrifugally. Most of the depressed fractures are over the frontoparietal region because the bone is thin and the specific location is prone to an assailant's attack.[7]

A depressed fracture may be open or closed. Open skull fractures, by definition, have a skin laceration over the fracture which runs through the paranasal sinuses and the middle ear structures, resulting in communication between the external environment and the cranial cavity. Open fractures may be clean or contaminated/dirty.[7]

Patients with depressed skull fractures present with a history of trauma, depression over their skull, neurological signs, seizure, and CSF leak; brain matter may come through the wound in compound depressed fracture (Fig. 1). A plain X-ray of the skull will demonstrate the fracture, type, its location, and its degree of depression. Computed tomography scan is helpful in the diagnosis of skull fracture and associated intracranial lesion. Generally, CT scan is more useful in demonstrating depressed fractures except when they are at the vertex.[8]

In the present series, there was male preponderance (male:female ratio 2.2:1), and road traffic accident was the common cause of depressed fracture followed by assault, which were similar to studies conducted elsewhere.[4,9,10]

Depressed skull fractures, a very serious type of trauma occurring in 11% of severe head injuries, account for significant morbidity and mortality,[1] and there is a consistent association between the presence of cranial fracture and higher incidence of intracranial lesions, neurological deficit, and poor outcome.[2] Cranial fracture is found to be the only independent significant risk factor in predicting intracranial hematomas.[11] In the present study, more than half of the patients had intracranial hematoma (extradural hematoma 50%, subdural hematoma 4.5%) (Fig. 5), which is lesser compared with the study done by Macpherson et al,[12] wherein 71% of the patients with cranial fracture had an intracranial lesion. Clinically, the chances of predicting the intracranial lesion by the presence of cranial fractures is further augmented by the presence of low GCS scores.[13,14] However, CT scan should be done in all cases for evaluation of patients with known or clinically suspected cranial fractures.

In the present series, nearly two-third of the cases had a compound (open) type (63.6%) of depressed fracture and the remaining had simple (close) type of depressed fracture, which was inconsonance with the study done by M Zahed Hossain et al[4] and less compared to other studies where compound fractures account for up to 90% of depressed fractures.[15-17]

Compound depressed skull fractures are surgical emergencies, and unless treated promptly and properly, complications like meningitis, cerebral abscess, osteomyelitis, or posttraumatic seizure may supervene.[5] However, there are two schools of thought in the management of depressed fractures. By convention, compound depressed cranial fractures are treated surgically, with debridement and elevation, primarily to attempt to decrease the incidence of infection. Closed or simple depressed cranial fractures undergo operative repair if the extent of depression is greater than the full thickness of the adjacent calvarium, with the theoretical benefits of better cosmesis, a diminution in late-onset posttraumatic epilepsy, and a reduction in the incidence of persistent neurological deficit. Some of them argue contrary to this conventional surgical management; in their studies they concluded that patients with open (compound) depressed cranial fractures may be treated nonoperatively if there is no clinical or radiographic evidence of dural penetration, significant intracranial hematoma, depression greater than 1 cm, frontal sinus involvement, gross deformity, wound infection, pneumocephalus, or gross wound contamination.[18,19]

In our series, for all patients, surgical management of the depressed fractures was done under the cover of prophylactic antibiotics and anticonvulsants by elevation

of depressed bone fragment, removal of in-driven bone fragment, repair of dural tear, evacuation of hematoma, hemostasis, debridement of wound margin, and primary repair.

The rationale behind aggressive treatment of depressed cranial fractures stems from their association with infection and late epilepsy. In our present series, postoperative wound infection was noted in 11.3% cases, which is comparable with other studies.[4,9] The postoperative incidence of wound infection was observed to be significantly associated with a significantly higher incidence of persistent neurological deficit, late epilepsy (defined as seizures longer than 1 week from injury), and death. The delay in operating the patient for more than 48 hours of injury dramatically increased the incidence to 36.5%. Operative debridement reduced the incidence of infection to 4.6% in their series.[20] Other complications in our series included meningitis (4.5%), CSF leak (9.1%), focal neurological deficits (6.8%), and pseudomeningocele (2.1%). Similar complications were reported by other studies.[4,16] All the complications were managed conservatively. Other complications reported by other studies include late epilepsy of up to 15% and mortality rate of 1.4 to 19%.[15,17,18,21]

CONCLUSION

Management of depressed fractures should be individualized depending upon the various important factors like degree of depression, communication with the exterior, neurological deficit, and presence of associated injuries.

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REFERENCES

1. Graham DI, Gennareli TA. Pathology of brain damage after head injury. In: Cooper P, Golfinos G, editors. Head injury. 4th ed. New York (NY): Morgan Hill; 2000.
2. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, Servadei F, Walters BC, Wilberger J, Surgical Management of Traumatic Brain Injury Author Group. Surgical management of depressed skull fractures. Neurosurgery 2006 Mar;58 (Suppl 3):52-60.
3. Rengachary SS, Ellenbogen RG. Principles of neurosurgery. In: Geisler FH, Mans PN, editors. Traumatic skull and facial fractures. 2nd ed. St Louis (MO): Elsevier; 2008. p. 329-335.
4. Hossain MZ, Mondle MS, Hoque MM. Depressed skull fracture: Outcome of surgical treatment. TAJ 2008;21(2):140-146.
5. Kalayanaraman S. Scalp and skull injuries. In: Ramamurthi B, Tandon PN, editors. Textbook of neurosurgery. New Delhi: Churchill Livingstone Pvt Ltd; 1996.
6. Gilbert S. Investigative significance of coup and contrecoup head injuries. 1969 [retrieved 2015 Aug 20]. Available from: eMedicine.com.
7. Qureshi NH, Harsh GR. Skull fractures. eMEDICINE, 2001. http://emedicine.medscape.com/article/248108-overview (Accessed on August 22, 2015).
8. Cowan BF, Segall HD, Zee CS, et al. Neuroradiological assesment of depressed skull fracture: axial versus skull roentgenography. Western Neuroradiological Society Annual Meeting; 1980 Oct.
9. Al-Haddad SA, Kirollos R. A 5-year study of the outcome of surgically treated depressed skull fractures. Ann R Coll Surg Engl 2002 May;84(3):196-200.
10. Ozer FD, Yurt A, Sucu HK, Tektaş S. Depressed fractures over cranial venous sinus. J Emerg Med 2005 Aug;29(2):137-139.
11. Chan KH, Mann KS, Yue CP, Fan YW, Cheung M. The significance of skull fracture in acute traumatic intracranial hematomas in adolescents: a prospective study. J Neurosurg 1990 Feb;72(2):189-194.
12. Macpherson BC, MacPherson P, Jennett B. CT evidence of intracranial contusion and haematoma in relation to the presence, site and type of skull fracture. Clin Radiol 1990 Nov;42(5):321-326.
13. Hung CC, Chiu WT, Lee LS, Lin LS, Shih CJ. Risk factors predicting surgically significant intracranial hematomas in patients with head injuries. J Formos Med Assoc 1996 Apr;95(4):294-297.
14. Servadei F, Ciucci G, Pagano F, Rebucci GG, Ariano M, Piazza G, Gaist G. Skull fracture as a risk factor of intracranial complications in minor head injuries: a prospective CT study in a series of 98 adult patients. J Neurol Neurosurg Psychiatry 1988 Apr;51(4):526-528.
15. Braakman R. Depressed skull fracture: data, treatment, and follow-up in 225 consecutive cases. J Neurol Neurosurg Psychiatry 1972 Jun;35(3):395-402.
16. Cooper PR. Skull fracture and traumatic cerebrospinal fluid fistulas. In: Cooper PR, editor. Head injury. 3rd ed. Baltimore (MD): Williams and Wilkins; 1993. p. 115-136.
17. Wylen EL, Willis BK, Nanda A. Infection rate with replacement of bone fragment in compound depressed skull fractures. Surg Neurol 1999 Apr;51(4):452-457.
18. Heary RF, Hunt CD, Krieger AJ, Schulder M, Vaid C. Nonsurgical treatment of compound depressed skull fractures. J Trauma 1993 Sep;35(3):441-447.
19. Van den Heever CM, van der Merwe DJ. Management of depressed skull fractures. Selective conservative management of nonmissile injuries. J Neurosurg 1989 Aug;71(2):186-190.
20. Jennett B, Miller J. Infection after depressed fracture of skull. Implications for management of nonmissile injuries. J Neurosurg 1972;36:333-339.
21. Colak A, Berker M, Ozcan OE. Occipital depression fractures in childhood. A report of 14 cases. Childs Nerv Syst 1991 Apr;7(2):103-105.